

**TRYPANOSOMA CRUZI: VESICLES SHED BY THE PARASITE GENERATE INTENSE INFLAMMATORY RESPONSE AND HIGHER NUMBER OF AMASTIGOTE NESTS IN INFECTED HEART TISSUE**

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Infective cultured-derived trypomastigotes of *T. cruzi* release into the culture medium vesicles enriched in glycoproteins of the gp85/Trans-sialidase superfamily and  $\alpha$ -galactosyl-containing glycoconjugates (Gonçalves et al., 1989). It was previously shown that an isolated fraction of the shed material, named Tc-Ves, induce potent inflammatory immune response and host cell invasion of macrophages *in vitro* (Torrecilhas et al, submitted). The effect of Tc-Ves *in vivo* was therefore investigated. BALB/c mice pretreated with Tc-Ves followed by trypomastigote infection show higher parasitemia and mortality when compared to the untreated animals. Moreover, Tc-Ves treated animals developed severe heart pathologies, with intense inflammatory reaction, higher number of intracellular amastigote nests and higher level of IL-10 mRNA and lower level of IFN- $\gamma$  transcripts when compared to the controls. The results indicate that vesicles released by *T. cruzi* trypomastigotes can modulate the infection *in vivo* by inducing IL-10 cytokine, and might play a role during the acute phase of the disease. Financial support: FAPESP and CNPq.